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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/943,780	08/30/2001	Kevin P. Baker	P2548P1C10	2570
7590	08/18/2008		EXAMINER	
Brinks Hofer Gilson & Lione P. O. Box 10395 Chicago, IL 60610			BLANCHARD, DAVID J	
			ART UNIT	PAPER NUMBER
			1643	
			MAIL DATE	DELIVERY MODE
			08/18/2008	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	09/943,780	BAKER ET AL.	
	Examiner	Art Unit	
	David J. Blanchard	1643	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1) Responsive to communication(s) filed on 12 May 2008.

2a) This action is **FINAL**. 2b) This action is non-final.

3) Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4) Claim(s) 27-29 and 32-34 is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5) Claim(s) _____ is/are allowed.

6) Claim(s) 27-29 and 32-34 is/are rejected.

7) Claim(s) _____ is/are objected to.

8) Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9) The specification is objected to by the Examiner.

10) The drawing(s) filed on _____ is/are: a) accepted or b) objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11) The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12) Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a) All b) Some * c) None of:

1. Certified copies of the priority documents have been received.
2. Certified copies of the priority documents have been received in Application No. _____.
3. Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) Notice of References Cited (PTO-892)

2) Notice of Draftsperson's Patent Drawing Review (PTO-948)

3) Information Disclosure Statement(s) (PTO/SB/08)
Paper No(s)/Mail Date _____.

4) Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____.

5) Notice of Informal Patent Application

6) Other: _____.

DETAILED ACTION

1. Claims 1-26, 30-31 and 35-36 are cancelled.
2. Claims 27-29 and 32-34 are pending and under consideration.
3. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Rejections Withdrawn

4. The rejection of claims 30-31 under 35 U.S.C. 101 because the claimed invention is not supported by either a credible, specific, and substantial asserted utility or a well established utility is withdrawn in view of the cancellation of the claims.
5. The rejection of claims 30-31 under 35 U.S.C. 112, first paragraph. Specifically, since the claimed invention is not supported by either a credible, specific, and substantial asserted utility or a well established utility for the reasons set forth above, one skilled in the art clearly would not know how to use the claimed invention is withdrawn in view of the cancellation of the claims.
6. The rejection of claims 27, 30-31 and 33-34 under 35 U.S.C. 112, second paragraph, as being indefinite in the recitation that the polypeptide comprises the "extracellular domain", optionally lacking its associated signal peptide is withdrawn in view of the amendments to the claims and the cancellation of claims 30-31.
7. The rejection of claims 27, 30-31 and 33-34 and are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement is withdrawn in view of the amendments to the claims and the cancellation of claims 30-31
8. The rejection of claims 30-31 under 35 U.S.C. 102(b) as being anticipated by Bostein et al (WO 99/35170, published 7/15/1999) is withdrawn in view of the cancellation of the claims.

Rejections Maintained

35 U.S.C. §§ 101 and 112, First Paragraph

35 U.S.C. 101 reads as follows:

Whoever invents or discovers any new and useful process, machine, manufacture, or composition of matter, or any new and useful improvement thereof, may obtain a patent therefor, subject to the conditions and requirements of this title.

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

9. The rejection of claims 27-29 and 32-34 under 35 U.S.C. 101 because the claimed invention is not supported by either a credible, specific, and substantial asserted utility or a well established utility is maintained.

The response filed 5/12/2008 states that the Office acknowledges that the nucleic acid encoding PRO357 is amplified in 93% of the lung tumor samples tested and that such data provides a sufficient utility with respect to a diagnostic in lung tumor tissue. Applicant maintains that it is more likely than not for amplified genes to have increased mRNA and protein levels because, in general, gene amplification increases mRNA expression and in turn, increased polypeptide levels. This has been fully considered but is not found persuasive. The *general* concept of gene amplification's lack of correlation with mRNA/protein overexpression in cancer tissue is addressed by Sen (2000, Curr. Opin. Oncol. 12:82-88). Specifically, Sen teaches that cancerous tissue is known to be aneuploid, that is, having an abnormal number of chromosomes. A slight amplification of a gene does not necessarily correlate with overexpression in a cancer tissue, but can merely be an indication that the cancer tissue is aneuploid. Furthermore, the art recognizes that lung epithelium is at risk for cellular damage due to direct exposure to environmental pollutants and carcinogens, which result in aneuploidy **before** the epithelial cells turn cancerous. See Hittelman (2001, Ann. N. Y. Acad. Sci. 952:1-12), who teach that damaged, precancerous lung epithelium is often aneuploid.

See especially p. 4, Figure 4. The gene amplification assay in the instant specification does not provide a comparison between the lung tumor samples and normal lung epithelium and does not correct for aneuploidy. Thus it is not clear that PRO357 is amplified in cancerous lung epithelium more than in damaged (non-cancerous) lung epithelium. One skilled in the art would not conclude that PRO357 is a diagnostic probe for lung cancer unless it is clear that PRO357 is amplified to a clearly greater extent in true lung tumor tissue relative to non-cancerous lung epithelium. Second, even if the data had been corrected for aneuploidy and a proper control had been used, the data have no bearing on the utility of the claimed PRO357 *polypeptides*. In order for PRO357 polypeptides to be overexpressed in tumors, amplified genomic DNA would have to correlate with increased mRNA levels and increased polypeptide levels. No data regarding PRO357 mRNA or PRO357 polypeptide levels in lung tumors have been brought forth on the record. The art discloses that a correlation between genomic DNA levels and mRNA levels cannot be presumed, nor can any correlation between genomic DNA levels and polypeptide levels, e.g., Pennica et al and Konopka et al of record. Further, Hanna and Mornin (1999, Pathology Associates Medical Laboratories) provide another important example of a lack of correlation between gene amplification and mRNA/polypeptide overexpression, wherein diagnosis of breast cancer included testing both the amplification of the HER-2/neu gene as well as over-expression of the HER-2/neu gene product. Thus, Hanna and Mornin provide evidence that the level of polypeptide expression must be tested empirically to determine whether or not the polypeptide can be used as a diagnostic marker for a cancer. The specification does not provide data as to whether or not the polypeptide level of PRO357 was tested in normal and cancerous tissue, and thus the skilled artisan *must* perform additional experiments, as directed by the art. Since the asserted utility for the claimed polypeptides is not in currently available form, and further experimentation is *required* to reasonably confirm the asserted real-world use, the asserted utility is not substantial.

Applicant states that to overcome the presumption of truth that Applicants' assertion of utility enjoys, the Office action must establish that it is more likely than not that one of ordinary skill in the art would doubt Applicants' assertion of utility. Applicant

relies upon Ornoff, Pollack, Hyman, Jares, Fan, Saretzki, Sohn, Forus, Walch, de la Guardia, Walker, Blancato, and Cancer medicine evidence that gene amplification more likely than not correlates with mRNA and polypeptide expression. At pp. 6-7 of the response Applicant refers to Orntoft et al, Hyman et al and Pollack et al as evidencing that, in general, gene amplification increases mRNA expression. This has been fully considered but is not found to be persuasive. Orntoft et al could only compare the levels of about 40 well-resolved and focused *abundant* proteins." (See abstract). It would appear that Applicants have provided no fact or evidence concerning a correlation between the specification's disclosure of *low* levels of amplification of DNA (which were not characterized on the basis of those in the Orntoft publication) and an associated rise in level of the encoded polypeptide. Hyman et al found 44% of *highly* amplified genes showed overexpression at the mRNA level, and 10.5% of *highly* overexpressed genes were amplified; thus, even at the level of high amplification and high overexpression, the two do not correlate. Further, the article at page 6244 states that of the 12,000 transcripts analyzed, a set of 270 was identified in which overexpression was attributable to gene amplification. This proportion is approximately 2%; the Examiner maintains that 2% does not provide a reasonable expectation that the slight amplification of PRO357 would be correlated with elevated levels of mRNA, much less polypeptide. Since Hyman et al found that less than half of the amplified genes were overexpressed at the mRNA level, Hyman et al supports the basis of the rejections that it is more likely than not that gene amplification *fails* to correlate with increased mRNA/polypeptide levels. Pollack et al is similarly limited to highly amplified genes which were not evaluated by the method of the instant specification. None of the three references are directed to gene amplification, mRNA levels, or polypeptide levels in lung cancer. Applicant states that Jares et al found a significant correlation between gene amplification and mRNA overexpression of PRAD-1 and Fan et al report similar results stating "we have shown that the hTERT oncogene is amplified in a significant proportion of medulloblastomas and other CNS embryonal neoplasms. This gene amplification correlates with increased expression of hTERT mRNA." Id at 1768. This has been fully considered but is not found persuasive. Jares et al also state that correlation between

PRAD-1 gene amplification and overexpression is difficult in solid tumors because of tumor heterogeneity and the possible dilutional effect of neoplastic tissue present in the samples (pg. 4816, 2nd col.). While Fan et al found a significant correlation between hTERT gene dosage and hTERT message level, Fan et al attempted to extend their studies of hTERT expression to the protein level using immunohistochemistry, and while the highly amplified medulloepithelioma was diffusely immunopositive for hTERT, in the other 13 cases, Fan et al found no correlation between DNA, RNA and protein levels (pg. 1768, 1st col.). Importantly, neither Jares et al nor Fan et al are directed to gene amplification, mRNA levels, or polypeptide levels in lung cancer, nor do Jares et al and Fan et al provide information on general trends in gene amplification, mRNA levels, or polypeptide levels in lung cancer.

Applicants' remarks regarding the supplied abstracts of Saretzki, Sohn, Forus, Walch, de la Guardia, Walker, Blancato, and Cancer medicine as providing additional evidence that gene amplification more likely than not correlates with mRNA and polypeptide expression are acknowledged, however, all of the evidence provided is directed to molecules that have been shown or are suggested to provide a selective growth advantage to the cell. It is not known whether PRO357 provides a selective growth advantage and further testing would be required in order to reasonably confirm such. Godbout et al., Sen, and Hittelman also speak to the general lack of correlation between gene amplification levels and protein expression for genes which are not known to provide a selective growth advantage to the cell. Additionally, while applicant only supplied the abstracts of Saretzki, Sohn, Forus, Walch, de la Guardia, Walker, Blancato, and Cancer medicine, Saretzki et al (Cancer Letters, 176:81-91, 2002) disclose that "A correlation between hTERT amplification and enhanced telomerase activity or higher hTERT protein levels has been shown recently in selected neuroblastoma cells and primary breast carcinoma, but could not be established in lung or cervical carcinomas." (pg. 90, 1st col.). Blancato et al (British Journal of Cancer 90:1612-1619, 2004) observed that in 40% of the high grade tumors tested, c-Myc protein was expressed at high levels, despite a lack of its gene amplification (pg. 1618, 2nd col., Table 5). De la Guardia et al (Head & Neck, 23:104-112, 2001) disclose that

36% of the tumors assessed for CENP-F mRNA overexpression (Tables 4 and 5) showed two- to fourfold overexpression of CENP-F mRNA. However, only 1 tumor had simultaneously CENP-F gene amplified and CENP-F mRNA overexpressed. This is not unusual, because frequent overexpression of c-MYC mRNA without c-MYC amplification and vice versa in various human tumors has been reported (pg. 111 1st col.). Accordingly, one skilled in the art would more likely than not doubt applicant's assertion that gene amplification more likely than not correlates with mRNA and polypeptide expression. None of the supplied references are directed to gene amplification, mRNA levels, or polypeptide levels in lung cancer.

The specification does not provide data as to whether or not the polypeptide level of PRO357 was tested in normal and cancerous tissue, and thus the skilled artisan *must* perform additional experiments, as directed by the art. Since significant further research would have been required of the skilled artisan to reasonably confirm that PRO357 polypeptides are overexpressed in any cancer to the extent that they could be used as cancer diagnostic agents, the asserted utility is not substantial. Even more research would be required of the skilled artisan to determine if the claimed PRO357 polypeptides can be used as cancer therapeutics, since there is no evidence that PRO357 plays a role in cancer formation or progression such that inhibiting PRO357 would result in effective cancer therapy. In the absence of information regarding whether or not PRO357 polypeptide levels are also different between specific cancerous and normal tissues, the proposed use of the PRO357 **polypeptides** as diagnostic markers and therapeutic targets are simply starting points for further research and investigation into potential practical uses of the claimed polypeptides.

See *Brenner v. Manson*, 148 U.S.P.Q. 689 (Sup. Ct., 1966), wherein the court held that:

"The basic quid pro quo contemplated by the Constitution and the Congress for granting a patent monopoly is the benefit derived by the public from an invention with substantial utility", "[u]nless and until a process is refined and developed to this point-where specific benefit exists in currently available form-there is insufficient justification for permitting an Appellant to engross what may prove to be a broad field", and "a patent is not a hunting license", "[i]t is not a reward for the search, but compensation for its successful

conclusion."

Applicants criticize Pennica et al as being limited to individual WISP genes, and that no general trends can be concluded therefrom. Applicants' point to the correlation between WISP-1 gene amplification and polypeptide overexpression. Applicants' also criticize Konopka et al. on the same grounds, i.e., that it is limited to a specific result and does not teach anything about gene amplification and protein over-expression in general. This has been fully considered but is not found to be persuasive. The instant application also presents data from a single gene at a time and makes conclusions about gene products from genomic DNA data. Pennica et al and Konopka et al constitute evidence that it cannot be assumed that amplified genomic DNA for a single gene results in overexpressed gene product. Godbout et al and Li et al also provide evidence to this effect with respect to the general concept of whether or not gene amplification correlates with increased mRNA/polypeptide expression. Finally, Sen and Hittelman (supra) constitute evidence that, in general, non-cancerous epithelial tissues are frequently aneuploid, and thus an increase in genomic DNA is not diagnostic of cancer. Applicants' arguments regarding U.S. Patent 7,208,308 are again acknowledged, however, Applicant is again reminded that each application is examined on its own merits, and the examiner is precluded from commenting on the '308 patent under 35 U.S.C. 282. It is noted that the '308 patent and the instant application do not claim the same subject matter, e.g., the polypeptide of SEQ ID NO:69.

At pp. 11-12 of the response, Applicants' take issue with the Godbout et al. reference, arguing that it was never claimed that PRO357 was similar to the DDX1 gene of Godbout et al. Appellants argue that Godbout et al show good correlation between gene amplification and protein expression levels. Applicants' assert that selective advantage to the cell is not the only mechanism by which genes impact cancer. Applicants' urge that structure/function data are not a requirement for utility. This has been fully considered but is not found to be persuasive. Applicants' assertions are in direct contradiction to the statements made in the Godbout et al evidence. Specifically, Godbout et al state that "***It is generally accepted that co-amplified genes are not***

over-expressed unless they provide a selective growth advantage to the cell.”

Applicants' have provided no evidence to contradict this. Godbout et al do show a good correlation between gene amplification and protein over-expression, but *only* when the gene provides a selective growth advantage to the cell. No evidence has been brought forward that PRO357 provides such an advantage.

At p. 12 of the response, Applicants' criticize Li et al. Applicants' urge that Li et al acknowledge that their results differed from those of Hyman et al and Pollack et al (of record), and note that the difference may be due to different methodologies. Applicants' refer to the supplemental information accompanying the Li et al article. Applicants' urge that Li et al. used an amplification copy ratio of only 1.40, which is not significant according to the Goddard declaration (of record) , and that a copy number of at least 2.0 was necessary. This has been fully considered but is not found to be persuasive. First, it is noted that Hyman et al also found that less than half of the amplified genes were overexpressed at the mRNA level, even though they only investigated genes in genomic DNA regions that were amplified at least 2-fold (argued in more detail above), and thus Hyman et al support the examiner's position. Furthermore, Li et al did not limit their studies to genes that were amplified at less than 2-fold. In fact, the supplemental information indicates that some of the samples were required to bind with a probe requiring at least 2-fold amplification:

Genes with copy number ratio > 1.40 (representing the upper 5% of the CGH ratios across all experiments) were considered to be overrepresented. A genomic fragment that contained six or more adjacent probes showing a copy number ratio > 1.40 , or a region with at least three adjacent probes with a copy number ratio > 1.40 **and no less than one probe with a ratio > 2.0** , were considered to be amplicons. (emphasis added, from 1st page of supplemental material)

Additionally, Li et al. clearly state: “***In our study, 68.8% of the genes showing over-representation in the genome did not show elevated transcript levels,*** implying that at least some of these genes are 'passenger' genes that are concurrently amplified because of their location with respect to amplicons but *lack biological relevance in terms of the development of lung adenocarcinoma.*” Since more than half of the amplified genes were not overexpressed, Li et al constitutes strong evidence that

it is more likely than not that gene amplification does NOT correlate with increased protein levels, absent evidence that the polypeptide has biological relevance in cancer. Appellants have not established that the genes in the 2-fold or more amplification level had correlation with protein over-expression whereas those between 1.4 and 2.0 lacked correlation. Godbout et al., Sen, and Hittelman also speak to the general lack of correlation between gene amplification levels and protein over-expression for genes which are not known to provide a selective growth advantage to the cell.

Applicants' position that it is more likely than not for amplified genes to have increased mRNA and protein levels because, in general, gene amplification increases mRNA expression and in turn, increased polypeptide levels is not found persuasive. Unlike the situations wherein a claimed compound has been tested and has shown a pharmacological activity and therefore has a therapeutic utility sufficient under the patent laws, or wherein an invention has only limited utility and is only operable in certain applications and therefore has some degree of utility sufficient for patentability, in the present situation Applicants have not provided any testing of PRO357 mRNA or PRO357 polypeptide expression. In the absence of any information on the role, activity or expression of the PRO357 polypeptide in cancer, the examiner therefore considers the asserted utilities to not be specific and substantial because the skilled artisan would not know if or how PRO357 polypeptide expression changes in cancer. Applicants' utility standard would mandate only a showing that it is "not implausible" that the invention will work for its intended purpose. If mere plausibility were the test for how to use a claimed invention, Applicants could obtain patent rights to "inventions" based on a disclosure consisting of little more than guesses as to the likelihood of their success. When one of the guesses later proved true, the "inventor" would be rewarded the spoils instead of the party who demonstrated that the method actually worked. A probable utility does not establish a practical utility, which is established by actual testing or where the utility can be "foretold with certainty." *Bindra v. Kelly*, 206 USPQ 570, 575 (Bd. Pat. Inter. 1979) (Reduction to practice was not established for an intermediate useful in the preparation of a second intermediate with a known utility in the preparation

of a pharmaceutical. The record established there was a high degree of probability of a successful preparation because one skilled in the art may have been motivated, in the sense of 35 U.S.C. 103, to prepare the second intermediate from the first intermediate. However, a strong probability of utility is not sufficient to establish practical utility.). Practical utility is a shorthand way of attributing “real-world” value to claimed subject matter. In other words, one skilled in the art can use a claimed discovery in a manner, which provides some immediate benefit to the public.

For these reasons, the rejection is maintained.

10. The rejection of claims 27-29 and 32-34 under 35 U.S.C. 102(b) as being anticipated by Bostein et al (WO 99/35170, published 7/15/1999) is maintained.

Applicants' maintain that the present application is entitled to the filing date of priority application 60/113,296, i.e., 12/22/1998, which discloses the PRO357 polypeptide and amino acid sequence as well as the gene amplification experiment described in Example 28 of the present specification is described in Example 2 of the '296 application. According to applicant, for the reasons discussed above in response to the rejections under 35 U.S.C 101 and 112 and applicants' previous arguments of record, the description of the gene amplification in the '296 application satisfies the utility and enablement requirements for the PRO357 polypeptide. This has been fully considered but is not found persuasive for the following reasons.

Under 35 U.S.C. 120, the claims in a U.S. application are entitled to the benefit of the filing date of an earlier filed U.S. application if the subject matter of the claim is disclosed in the manner provided by 35 U.S.C. 112, first paragraph in the earlier filed application. Under 35 U.S.C. 119 (a) or (e), the claims in a U.S. application are entitled to the benefit of a foreign priority date or the filing date of a provisional application if the corresponding foreign application or provisional application supports the claims in the manner required by 35 U.S.C. 112, first paragraph. A deficiency under 35 U.S.C. 101 also creates a deficiency under 35 U.S.C. 112, first paragraph. In view of the deficiencies under 35 U.S.C. §§ 101 and 112, first paragraph set forth above, the claims

are not entitled to the benefit of the filing date of the earlier filed applications. Accordingly, the effective filing date for the claimed invention is 8/30/2001, which is the filing date of the instant application and the rejection is maintained.

11. No claim is allowed.
12. **THIS ACTION IS MADE FINAL.** Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to David J. Blanchard whose telephone number is (571) 272-0827. The examiner can normally be reached at Monday through Friday from 8:00 AM to 6:00 PM, with alternate Fridays off. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Larry Helms, can be reached at (571) 272-0832.

The official fax number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you

have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/David J. Blanchard/
Primary Examiner, A.U. 1643